Role of Microsclerotia as Primary Inoculum of *Microdochium panattonianum*, Incitant of Lettuce Anthracnose

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ABSTRACT

Patterson, C. L., and Grogan, R. G. 1991. Role of microsclerotia as primary inoculum of *Microdochium panattonianum*, incitant of lettuce anthracnose. Plant Dis. 75:134-138.

Microdochium panattonianum, incitant of lettuce anthracnose, was soilborne. Lettuce planted in soil collected from fields 1–4 yr after an epidemic, or in soil artificially contaminated with residue infected with *M. panattonianum*, developed anthracnose. The pathogen was eliminated from soil, however, by fumigation with methyl bromide. Microsclerotia were observed in cells of lettuce tissues infected by *M. panattonianum*. Microsclerotia also were produced in vitro on a basal salts agar medium, germinated, infected, and incited anthracnose on inoculated lettuce; optimum conditions for germination and infection were 6 hr of continuous leaf wetness at 20–22 C. Anthracnose occurred on lower leaves of lettuce planted in soil contaminated with microsclerotia placed at 0-, 1-, or 2-cm depths. Thus, microsclerotia produced by *M. panattonianum* in infected lettuce residue were a primary source of soilborne inoculum that initiated anthracnose epidemics.

Lettuce (*Lactuca sativa* L.) anthracnose is caused by *Microdochium panattonianum* (Berl.) Sutton, Galea & Price (6) (= *Marssonina panattoniana* (Berl.) Magnus). In addition to cultivated lettuce, the pathogen infects several *Lactuca* weed species (1,2,8,10).

Anthracnose usually is first evident on lower leaves of lettuce near or in contact with soil (1,2,4,5,8,10,12). Symptoms are small (0.5–1.0 mm), circular, chlorotic lesions that become pink during sporulation. Leaf lesions become necrotic, and infected tissues disintegrate to produce shot-hole symptoms (10). Midrib and petiole lesions elongate and appear pitted; the infected tissues, however, remain intact. During periods of frequent rainfall and cool temperatures optimum for *M. panattonianum* (1,2,4), infected plants may defoliate, thus decreasing quality and salability at the market (1,2, 4,5,8–10,12).

Stevenson (12) proposed that primary inoculum of *M. panattonianum* was seedborne. She and Couch and Grogan (2) reported that conidia survived on artificially contaminated lettuce seed for 4 days. Galea and Price (5) demonstrated that transmission was significantly reduced 5 days after seed were contaminated with conidia and nil after 24 days in storage. More important, however, seed harvested from Cos Verdi lettuce infected with *M. panattonianum* did not transmit the pathogen to germinating seedlings. Commercial lettuce seed is usually produced in a hot, dry climate suboptimal to the disease cycle of *M. panattonianum* (2,4) and development of anthracnose (1,2,4). Thus, seedborne conidia likely were not a means of survival, transmission, or dispersal of *M. panattonianum*.

Newhall (8) suspected that *M. panattonianum* overwintered in stem lesions on residues of *Lactuca* weed species. Couch and Grogan (2) incited anthracnose symptoms on cultivated lettuce by inoculating plants with a water homogenate of quiescent lesions of *M. panattonianum* obtained from *L. serriola* f. *intergrifolia* Bogenh. Most lettuce producers in California effectively control weeds to eliminate alternate host reservoirs of lettuce mosaic and other virus diseases (10). In addition, anthracnose epidemics occurred in the Salinas Valley when weeds infected with *M. panattonianum* were not growing in the vicinity of affected fields (9,10). In the rare event infected weeds were identified, disease gradients from field borders were not observed. It was, therefore, unlikely that alternate *Lactuca* hosts infected by *M. panattonianum* contributed sufficient inoculum to initiate anthracnose epidemics.

Numerous reports (1,2,4,5,8–10) associated anthracnose epidemics with plantings in fields previously cropped to lettuce infected by *M. panattonianum*. Several assays were developed to detect the pathogen in contaminated fields (2,5,8). The fungus was reported to persist in soil-incorporated infected lettuce residue for one to several months (1,2,5,8) and 58 mo in residue that was not associated with soil (5). Conidia, however, were short-lived and survived in artificially contaminated soil about 10 wk (5). Differences in inoculum longevity were attributed to soil type (5), soil microclimate (1,2,5,8,12), rate of residue decomposition (2,5), and fungistasis attributable to unidentified compounds in soil (5).

In the Salinas Valley, lettuce anthracnose epidemics occurred during wet springs after prolonged periods (up to 5 yr) of drought, fallow, and/or rotation with nonhost crops (2,9,10). The disease commonly occurred in randomly distributed foci of one to several plants (2,10). In several fields, the duration of rotation out of lettuce infected with *M. panattonianum* was greater than the reported longevity of the pathogen in soil (1,2,5,8). Previous research (1,2,5,8,9) and observations (1,10,12) presented convincing evidence that the primary inoculum of *M. panattonianum* might be soilborne. There was discrepancy, however, concerning the etiology (1,2,5,6,8–10,12) and longevity (1,2,5,8) of inoculum in soil. Microsclerotia were observed in cells of lettuce infected by *M. panattonianum* before the disintegration of necrotic tissues (9,10). The propagules also were produced in vitro on a basal salts agar medium, and anthracnose lesions developed 5–7 days after lettuce was inoculated with the microsclerotia (9). Thus, microsclerotia likely were responsible for the persistence of *M. panattonianum* in soil during drought, fallow, and rotation with unsusceptible crops.

The purpose of this research was to: 1) confirm the association of anthracnose epidemics with previous plantings of lettuce infected by *M. panattonianum*, 2) determine the longevity of *M. panattonianum* in soils after rotation with nonhost crops, 3) establish the importance of microsclerotia as primary inoculum, 4) identify optimum conditions for germination and infection of lettuce by microsclerotia, and 5) assess inoculum placement in soil relative to infection of lettuce.

MATERIALS AND METHODS

Ten isolates of *M. panattonianum* were obtained from lettuce leaf lesions incubated on potato-dextrose agar (PDA) (3) acidified to about pH 4.5 with 25% lactic acid (APDA). Single-spore cultures were derived by inocu-

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This research was funded in part by the California Iceberg Lettuce Research Board.

Accepted for publication 3 July 1990 (submitted for electronic processing).

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lating APDA plates with 0.1 ml of about 10^3 conidia per milliliter of sterile distilled water. A hyphal tip from single-spore colonies was transferred and maintained on PDA slants stored at 5 C.

Because previous observations and inoculation studies assessed Paris Island Cos lettuce as highly susceptible to anthracnose (Patterson and Grogan, *unpublished*), the cultivar was used in all tests. Experiments addressing soilborne potential of *M. panattonianum* were conducted with potted soil on benches in fields located at the University of California Plant Pathology farm in Davis. Daily observations for anthracnose continued 21 days after the first visible disease symptoms (symptoms evident after 21 days were considered to result from infections by conidia produced elsewhere). Treatments were replicated in a randomized complete block design (11). Data were tested by single or multifactorial analysis of variance (ANOVA) (11), determined by the number of factors assessed. Significant differences in treatment means were identified by Fisher's protected least significant difference test (PLSD) (11) at the 99% confidence level ($P = 0.01$). Other analyses were included when appropriate and referenced in materials and methods of specific experiments. Data were presented as the mean of factors assessed.

Longevity of *M. panattonianum* in soil after rotation with nonhost crops. The longevity of *M. panattonianum* in contaminated fields was determined by randomly collecting 500 soil samples 8 × 20 cm (diameter × depth = approximately 1,000 cc) from fields in the Salinas Valley 1–5 yr after an anthracnose epidemic. Each year of rotation was represented by samples from three fields for a total of 15 soils assayed (Table 1). Rotation crops included: celery (*Apium graveolens* L. var. *dulce* (Mill.) Pers.), various cole crops (*Brassica* sp.), spinach (*Spinacia oleracea* L.), carrots (*Daucus carota* L. subsp. *sativus* (Hoffm.) Arcang.), and onion and garlic (*Allium* sp.). Soil samples were bulked in reference to field location, thoroughly mixed, and potted in 10 × 15 cm (diameter × depth = approximately 1,180 cc) containers. Lettuce seed was planted and thinned to three plants per pot spaced about 5 cm apart in a triangular configuration. The soil treatments were replicated four times and included 25 pots per replicate. Pots within a replicate were spaced 30 cm apart, and replicates were separated by 3 m. Nearly all fields previously cropped with lettuce potentially were contaminated with inoculum of *M. panattonianum*. Controls to assure that infections were caused by soilborne inoculum and were not the result of nonpoint sources of conidia were, therefore, collected from an equivalent number of fields with no cropping history of lettuce. The potted control soils were planted with lettuce and randomly placed in the experimental block to identify infections from conidia (controls are not identified in Table 1).

Soil fumigation with methyl bromide. Soil was randomly collected as previously described from four fields in the Salinas Valley 1 mo after disking lettuce residue infected by *M. panattonianum*. About 300 L of soil from each field was fumigated with 454 g of methyl bromide in individual sealed 500-L metal containers. Four days after fumigation, the containers were opened and the methyl bromide was allowed to dissipate for 14 days. The respective fumigated and unfumigated soils were potted in 1-L samples replicated four times with 25 samples per replicate. The soils were irrigated and planted with lettuce (one plant per pot) 14 days later. The test was repeated one time.

Soil incorporation of lettuce residue infected with *M. panattonianum*. Lettuce was direct-seeded in 1 L of steam-sterilized Yolo fine sandy loam soil and thinned to one plant per pot. In the evening during a 4- to 8-hr dew period favorable for infection (2,4), 100 plants in the four-true-leaf stage were inoculated to runoff with about 0.5 ml of a suspension with 10^3 conidia of *M. panattonianum* per milliliter in sterile distilled water. Controls were represented by 100 plants treated with an equal volume of sterile distilled water. Cross-contamination was prevented by separating inoculated and uninoculated plants about 5 km. As *M. panattonianum* lesions developed, infected tissues were microscopically examined for microsclerotia. During March, about 4 mo after planting, infected and uninfected residues were incorporated into the respective potted soils and allowed to decompose for 8 mo before replanting. The soils were replanted three times during a 3-yr period; infections from soilborne inoculum were assessed by the occurrence of anthracnose in each successive planting.

In vitro production and temperature effects on germination of microsclerotia. Microsclerotia of 10 isolates of *M. panattonianum* were produced in vitro on a basal salts agar medium (BSAM) containing (per liter of distilled water) 680 mg of K_2HPO_4, 180 mg of $MgSO_4$, 149 mg of KCl, 18.4 mg of $MnSO_4$, 11.2 mg of $ZnSO_4$, 4.0 mg of $CuSO_4$, 4.0 mg of $FeCl_3$, 18 g of D-glucose, 12 g of Noble agar (Difco), and L-asparagine as the nitrogen source (equivalent of 0.5% actual N). A 5-mm-diameter disk of actively growing hyphae was extracted from 96-hr PDA cultures of the pathogen and aseptically transferred to about 15 ml of BSAM in 100 × 15 mm petri dishes. The cultures were incubated at 21 C and periodically examined for microsclerotia. After 21 days of growth and production of microsclerotia, the medium was gently washed for 24 hr in running tap water to dislodge conidia. The cultures were incubated an additional 24 hr at 40 C under forced air to desiccate remaining conidia and hyphae by dehydrating the medium. Dehydrated cultures were pulverized in a 500-mesh Wiley mill, suspended in 0.2% Tween 20, comminuted in a homogenizer for 5 min, and passed through Whatman No. 1 filter paper to remove agar debris from the suspension of microsclerotia. The filtrate was adjusted to 10^3 microsclerotia per milliliter and applied via an atomizer to microscope slides coated with a thin layer of 2% Bacto agar (Difco). Individual slides were placed in petri dishes and incubated for 72 hr at 100% RH, 12 hr diurnal light, at temperatures ranging from 4 to 40 C (in 2-C increments). After incubation, 10 slide replicates of each isolate and temperature regime were stained with 0.1% cotton blue in lactophenol to terminate germination and facilitate observation of microsclerotia.

Percentage of germination was determined by microscopic examination of 100 microsclerotia per slide. Factors assessed included the isolate of *M. panattonianum*, temperature regime, and germination. In addition to ANOVA and PLSD, the level of influence temperature (independent variable) had on germination (dependent variable) was determined by regression analysis (11).

Minimum duration of leaf wetness, temperature effects, and optimum conditions for infection of lettuce by microsclerotia. Lettuce was planted in 100 ml of U.C. growing medium contained in

Table 1. Longevity of *Microdochium panattonianum* in soil after rotation with nonsusceptible crops in Salinas Valley, California

Soil sample[a]	Years out of infected lettuce[b]	Infected plants[c] (%)
1	1	100
2	1	100
3	1	100
4	2	100
5	2	92
6	2	83
7	3	90
8	3	79
9	3	91
10	4	35
11	4	22
12	4	26
13	5	0
14	5	0
15	5	0

[a] Bulk composite of 500 soil samples from each field.
[b] Rotation crops did not significantly ($P = 0.01$) influence longevity of inoculum of *M. panattonianum* in soil. Anthracnose did not occur on lettuce planted in control soils randomly placed in the experimental block to identify infection from nonpoint sources of conidia.
[c] Average of 25 plants in each of four replicates. PLSD (0.01) = 48.37 (differences in percentage of infection).

5 × 5 × 5 cm (125-cc) pots (one plant per pot). Plants in the four-true-leaf stage were inoculated with about 0.5 ml of sterile distilled water with a concentration of 10^3 microsclerotia of *M. panattonianum* per milliliter. Comparative treatments included plants inoculated with equivalent concentrations of desiccated hyphae (forced air homogenized in sterile distilled water; each hyphal fragment represented a potential infective propagule) or desiccated conidia (dehydrated 24 hr before suspension in sterile distilled water). Controls were uninoculated plants. The desired duration of leaf wetness was maintained by placing inoculated plants in clear 1-mil plastic bags. Plants were incubated in growth chambers with temperatures ranging from 4 to 40 C (in 2-C increments) and 12 hr diurnal light. After 0, 2, 4, 6, 8, 10, 12, 24, 48, or 72 hr of leaf wetness, the plants were removed from the plastic bags, rapidly dried at 20 C under forced air, and placed in a greenhouse environment favorable to the development of anthracnose (2,4). Five plant replicates were included for each isolate, duration of leaf wetness, and temperature regime.

Fourteen days after plants were removed from growth chambers, the number of anthracnose lesions per inoculated leaf was recorded. Factors assessed included the isolate of *M. panattonianum* tested, duration of leaf wetness, and temperature regime. The level of influence that leaf wetness (independent variable, X_1) and temperature (independent variable, X_2) had on infection (dependent variable, Y) was determined by multiple regression analysis (11).

Soil placement of microsclerotia relative to infection of lettuce. Inoculum of one isolate of *M. panattonianum* was prepared by mixing microsclerotia with steam-sterilized Yolo fine sandy loam soil to a final concentration of about 10^3 propagules per gram. Five grams of the inoculum were evenly dispersed at respective depths ranging from 0 to 10 cm (1-cm increment per treatment) in the profile of 1 L of potted steam-sterilized Yolo fine sandy loam soil. Comparative inoculum treatments were equivalent concentrations of desiccated hyphal fragments or desiccated conidia and were also mixed with soil and placed at indicated soil depths. Uncontaminated soil was used for control treatments. Each inoculum treatment and depth of placement was prepared in individual pots containing three lettuce plants and replicated five times. Pots within a replicate were spaced 45 cm apart, and replicates were separated by 3 m. The experiment was repeated two times. Factors assessed included type of inoculum, depth of soil placement, and number of tests.

RESULTS

Longevity of *M. panattonianum*. Anthracnose was not evident on lettuce grown in control soils. In addition, rotation crops did not significantly ($P = 0.01$) influence the longevity of *M. panattonianum* in soil.

There were no significant differences ($P = 0.01$) in the percentage of infected plants when lettuce was grown in soil collected 1–3 yr after an anthracnose epidemic (Table 1). Mean infection of plants grown in three soils previously cropped with lettuce 1 yr after an anthracnose epidemic was 100%. Anthracnose developed on 92% of the plants grown in three soils collected from fields 2 yr after an epidemic. *M. panattonianum* infected 87% of the plants grown in soils collected 3 yr after an anthracnose epidemic. Percentage of infection significantly decreased ($P = 0.01$) when lettuce was grown in soils collected 4 yr after an epidemic. Anthracnose symptoms were not evident on plants grown in soil 5 yr after an epidemic.

Soil fumigation. Anthracnose did not develop on lettuce grown in soils fumigated with methyl bromide. In nonfumigated soils, 88–96% of the plants were infected by *M. panattonianum*.

Incorporation of lettuce residue infected with *M. panattonianum*. Microsclerotia were evident in the cells of lettuce tissues infected by *M. panattonianum* (Fig. 1) before soil incorporation of residue. Anthracnose occurred in three successive plantings of lettuce in soil contaminated with infected residue. The disease did not develop, however, when lettuce was grown in soil contaminated with noninfected residue.

Production and germination of microsclerotia. Numerous microsclerotia were produced by all isolates of *M. panattonianum* after 21 days of incubation on BSAM. The propagules resulted from differentiation of individual hyphal cells to form prosenchyma tissues (7), were slightly pigmented, and were 30–60 μm in diameter.

Microsclerotia germinated by production of hyphae that, in turn, gave rise to conidia (Fig. 2). Germination occurred at temperatures ranging from 10 to 30 C (Fig. 3) and was optimum at 20–22 C ($P = 0.01$). The influence of temperature on germination of microsclerotia was highly significant ($Y = 0.85$) and expressed as a quadratic equation (11) illustrated by the curve represented in a second-degree polynomial (Fig. 3) (11).

Temperature and leaf wetness. Anthracnose did not develop on lettuce inoculated with hyphae or conidia. A minimum duration of 4 hr of leaf wetness was required for infection of lettuce by microsclerotia (Table 2). Lesions per leaf significantly increased ($P = 0.01$) as leaf wetness increased to 6 hr; no significant increases in lesions per leaf were attributable to durations of leaf wetness greater than 6 hr. Anthracnose was not evident on lettuce incubated at temperatures below 10 C or above 30 C. Lesions per leaf significantly increased ($P = 0.01$) as the temperature increased from 10 to 20 C and decreased as the temperature increased from 24 to 30 C.

The influence of leaf wetness (X_1) and temperature (X_2) on germination and infection of lettuce by microsclerotia was highly significant ($Y_{X1X2} = 0.89$). Optimum conditions ($P = 0.01$) for infection were a minimum duration of 6 hr of continuous leaf wetness at 20–22 C.

Inoculum placement relative to infection. Anthracnose occurred on all plants grown in soil contaminated with microsclerotia placed at 0 and 1 cm. Disease incidence significantly decreased ($P = 0.01$) to 15% of plants infected when inoculum was placed at 2 cm. Anthracnose did not develop on lettuce when microsclerotia were placed below 2 cm or on

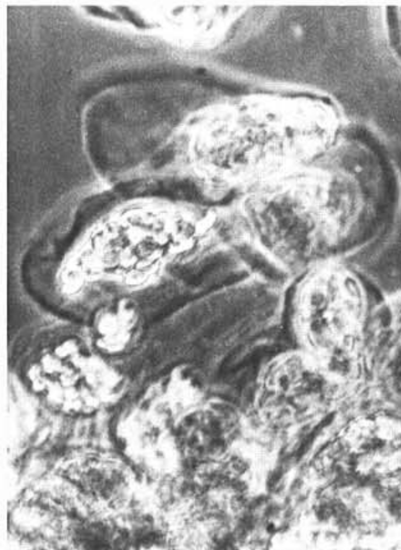

Fig. 1. Microsclerotia of *Microdochium panattonianum* within cells of infected lettuce tissues. (×400)

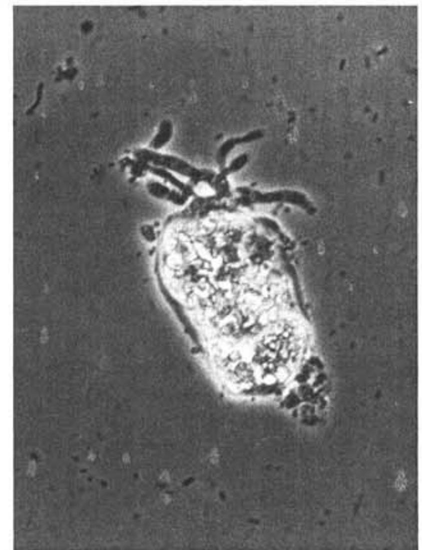

Fig. 2. Germination of a microsclerotium of *Microdochium panattonianum* produced in vitro on agar media. (×400)

plants grown in noncontaminated soil, or in soil contaminated with hyphae or conidia.

DISCUSSION

Development of anthracnose on lettuce grown in soils collected from fields 1–4 yr after an epidemic, and elimination of the pathogen by soil fumigation with methyl bromide, supported previous reports (1,2,4,5,8–10,12) that the primary inoculum of *M. panattonianum* can be soilborne. Although contrary to previous reports (1,2,5,8), data concerning longevity of the pathogen indicated inoculum persisted in Salinas Valley soils about 4 yr without a susceptible *Lactuca* host. Long-term residency of soilborne inoculum was, therefore, responsible for the repeated occurrence of anthracnose epidemics in California after extended periods of drought and/or rotation out of lettuce.

The occurrence of anthracnose on lettuce grown in soil artificially contaminated with residue infected with *M. panattonianum* in this and other studies (2,5,8) presented substantial evidence to conclude that the pathogen was harbored in debris. Soilborne inoculum, however, was not described in previous reports (1,2,5,8,12) concerning the longevity of *M. panattonianum* in soil. Further, Galea et al (6) did not allude to the etiology of soilborne inoculum in recent taxonomic reclassification of the pathogen. Nevertheless, microsclerotia of *M. panattonianum* were previously described (9,10). In the current study, the propagules were consistently evident in cells of lettuce tissues infected with *M. panattonianum*, and anthracnose occurred on plantings in soil contaminated with infected residue. In addition, Koch's postulates in this and a previous study (9) demonstrated that microsclerotia produced by the pathogen in vitro were capable of infecting lettuce. Thus, microsclerotia were an important source of soilborne primary inoculum initiating lettuce anthracnose epidemics caused by *M. panattonianum*.

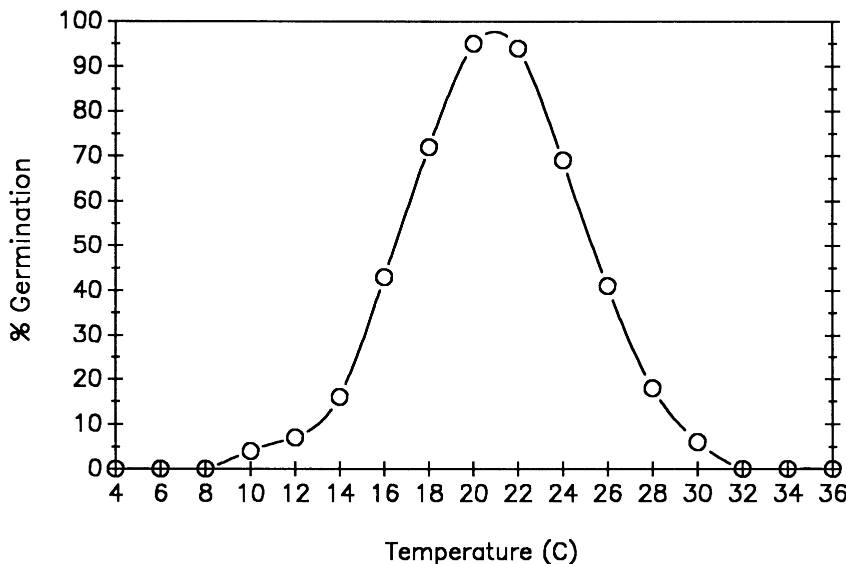

Fig. 3. Effect of temperature on germination by microsclerotia of *Microdochium panattonianum*. Average of 10 isolates in two experiments replicated 10 times. PLSD (0.01) = 17.41, Y = 0.85.

Table 2. Effect of duration of leaf wetness and temperature on infection of Paris Island Cos lettuce by microsclerotia of *Microdochium panattonianum*

Temperature (C)	Lesions per leaf at indicted hours of leaf wetness[a]									
	0	2	4	6	8	10	12	24	48	72
4	0	0	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0	0	0
10	0	0	2	6	7	6	8	7	7	6
12	0	0	6	9	9	8	9	8	9	9
14	0	0	7	13	13	14	13	14	14	14
16	0	0	12	16	15	16	17	15	16	15
18	0	0	17	21	22	20	22	22	21	22
20	0	0	24	29	30	30	29	31	30	29
22	0	0	25	30	30	29	30	32	30	31
24	0	0	18	22	23	21	23	22	23	23
26	0	0	13	16	15	16	17	15	16	16
28	0	0	7	10	9	11	10	9	11	11
30	0	0	3	6	6	6	7	6	6	7
32	0	0	0	0	0	0	0	0	0	0
34	0	0	0	0	0	0	0	0	0	0
36	0	0	0	0	0	0	0	0	0	0
38	0	0	0	0	0	0	0	0	0	0
40	0	0	0	0	0	0	0	0	0	0

[a] Average of four leaves per five plants per five isolates in two experiments. PLSD (0.01) = 2.79. $Y_{X1X2} = 0.89$.

Temperature range (10–30 C), duration of leaf wetness (minimum of 4 hr), and optimum conditions (≥6 hr of leaf wetness at 20–22 C) for germination of microsclerotia and infection of lettuce by *M. panattonianum* were different from requirements for germination and infection by conidia (2,4). Environmental conditions favorable for development of anthracnose epidemics may, therefore, differ from those optimum for disease progression. Because anthracnose epidemics in California occurred after prolonged periods of drought and/or rotation out of lettuce, microsclerotia likely can survive long periods of hot, dry weather. In addition, epidemics occurred only during years of high rainfall. Thus, activation and germination of microsclerotia may require longer durations of leaf wetness than reported in this study, possibly dependent on the length of time the propagules were dormant in soil. In lieu of data presented, however, leaf wetness was the limiting factor ($P = 0.01$) in germination of microsclerotia.

It was uncertain how lettuce plants were inoculated by soilborne microsclerotia of *M. panattonianum* in contaminated fields. Anthracnose symptoms usually were first evident on lower leaves near the soil surface (1,2,4,5,8,10,12). Thus, it was possible that microsclerotia were deposited on leaves in splashing soil dispersed by rain or sprinkler irrigation. When conditions were favorable, the microsclerotia germinated, infected, and initiated anthracnose epidemics.

The maximum depth of microsclerotia in soil relative to infection of lettuce may depend, in part, on rainfall velocity, impact on soil, and consequent soil dispersal in splashing water. In our study, only those microsclerotia in the upper 2 cm of soil were dispersed to inoculate and infect lettuce, as evidenced by disease development. Thus, application of fungicides to the soil or elimination of lettuce residue infected with *M. panattonianum* from the soil surface by plowing or other means of sanitation may result in short-term control of anthracnose.

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